Immunoglobulin IgA

Serum IgA is absent at birth but appears at about four weeks of age and by the age of 12 months is near the adult level. The serum values in children range from 49 to 114 mg per 100 ml. Serum IgA has a molecular weight of 165,000 and had a 7S sedimentation coefficient. Secretory IgA is similar to serum IgA. It is present in secretions in pairs linked to a "secretory piece." This combination is thought to be made locally in mucous membranes. "Secretory piece" is a G-globulin with a molecular weight of 50,000. Theories suggest that local antibodies, especially IgA, are important in the resistance to respiratory tract infections and play an important role in gastrointestinal and genitourinary tracts. Serum IgA is present in parotid, bronchial, small intestinal, prostatic and vaginal secretions as well as in colostrum, amniotic and lachrymal fluids.

M. MILLMAN, M.D.

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Lewis DM, Lapp NL, Burrell RR: Quantitation of secretory, immunoglobulin A in chronic pulmonary disease. Amer Rev Resp Dis 101:55-61, 1970

New Information on Allergic Rhinitis

Important information on the mechanism producing allergic nasal symptoms has emerged from studies made possible by the development of an instrument for measuring the effective nasal airway. These measurements have been obtained in conjunction with a method permitting control of the rate and amount of pollen administered intranasally. Objective responses have been measured quantitatively, under controlled conditions, before, during and after therapy.

The parameters of the nonspecific primary effect have been defined. An increase in reactivity of the nasal mucus membrane following repeated exposure to pollen is only slowly reversible over a period of days to weeks. By administering pollen to one nostril, this was shown to be a local effect rather than systemic. This resulted in unilateral priming and allergic rhinitis in the challenged nostril only.

Priming has been shown to be nonspecific in that hyperreactivity induced by one pollen (to which the patient is sensitive) results in a pronounced increase in sensitivity to a low dosage of another, unrelated pollen. This finding reemphasizes the importance of considering "the total allergic load" when evaluating allergic reactions.

Recently attempts to suppress the nasal membrane's allergic reaction to pollen by a nasal spray containing blocking antibody has had some success. Thus, it may be possible to treat patients with allergic rhinitis by first stimulating blocking antibody synthesis by conventional injection of antigen and then using the serum as a source of blocking antibody for use in nasal sprays.

JAMES P. KEMP, M.D.
ROBERT N. HAMBURGER, M.D.

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Hypersensitivity to Organic Dusts

An increasing number of organic dusts have been shown to produce allergic lung diseases similar to farmer's lung. Persons exposed develop precipitins which react specifically with antigens in the dust. Inhalation of the dust apparently incites an arthus reaction in the lung. Alveolitis and pulmonary fibrosis follow. About half the patients have repeated bouts of fever and pneumonitis. The remainder have a slowly progressive course, with cough, weight loss and pulmonary infiltration. Coexisting reaginic (IgE-mediated) allergy, as revealed by immediate wheal and and erythema skin tests, may modify the symptom pattern to one of asthma plus pulmonary infiltration.

A partial list of these diseases and the dusts which cause them: *Farmer's lung*—moldy overheated hay; *bagassosis*—moldy sugarcane bagasse; *maple bark, sequoia bark, oak bark pneumonitis*—moldy bark; *bird breeder's lung*—pigeon and budgerigar droppings. New diseases of this type are being found. The newest is *washing powder*

worker's lung—due to the enzymes from B. subtilis in washing powders.

Diagnosis is made by history, the presence of precipitins in the serum and a delayed, arthus-like reaction on skin testing. Treatment with adrenal corticosteroids seems to help, but avoidance of the offending dust is the most effective therapy.

GILDON N. BEALL, M.D.

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Pepys J: Hypersensitivity Diseases of the Lungs Due to Fungi and Organic Dusts—IV. Monographs in Allergy. Basel, Karger, 1969

The Beta-Adrenergic Blockade Hypothesis of Asthma

Beta-adrenergic stimulators, such as epinephrine and isoproterenol, relax bronchial smooth muscle, decrease glandular secretion, constrict blood vessels and also alleviate asthma. Beta-adrenergic blockers, such as propranolol (Indreal®), have the opposite effects. These observations have led to the theory that the basic defect in asthma is a partial β-adrenergic blockade. The causes of this postulated blockade might include hereditary influences and infections. Although as yet unproven, this theory has stimulated much new thought and experimental work on asthma. The theory would explain the diminishing therapeutic effectiveness of beta stimulators in patients with long-standing asthma and the subnormal cardiovascular and metabolic responses of asthmatic patients to β-adrenergic stimulators. On the other hand, normal subjects given propranolol do not develop the bronchial sensitivity so characteristic of asthma, and exercise-induced asthma seems to occur via mechanisms other than β-adrenergic blockade. Additionally, new information suggests that decreased β-adrenergic (epinephrine) secretion by asthmatics during stress may be related to their bronchial hyperreactivity.

GILDON N. BEALL, M.D.

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Reed C: β-adrenergic blockade in bronchial asthma and atopy. J Allergy 42:238-242, 1968

Hereditary Angio-Edema

Urticaria and angio-edema are common complaints. Foods, inhalants, insect stings, intestinal parasites and medications are frequent causes. There are other factors, some simple and some complex, such as: solar, thermal, pressure, infections and neoplasms.

Thompson gave an excellent review of these mechanisms which produce lesions by release of histamine.

Donaldson and Evans showed that patients with hereditary angio-edema are lacking alpha globulin which inhibits the esterase activity of complement.

Patients with hereditary urticaria and angio-edema need a confirmed laboratory diagnosis, as surveys show a 28 percent death rate from laryngeal edema. Antihistamines and steroids reduce attacks, but an adequate airway with epinephrine or isoproterenol are necessary in life-threatening attacks.

JOHN S. O'TOOLE, M.D.

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Thorvaldsson SE, Sedlack RE, Gleisch GJ, et al: Angioneurotic edema and deficiency of C:1 esterase inhibitor in a 61-year-old woman. Ann Intern Med 71:353-357, 1969

Newer Trends in Corticosteroid Therapy

A dosage schedule for corticosteroids involving the administration of the total 24-hour dosage in a single dose every other day rather than in divided doses during this same period has been fairly well accepted. Evidence of satisfactory efficacy and of substantially reduced side effects with long-term alternate-day therapy has been well documented.

It has been recommended that any patient requiring prolonged corticosteroid administration be given a trial of alternate-day therapy before being committed to a long term daily corticosteroid regime. Patients who require daily therapy at the onset to control symptoms should have regular attempts made to switch to an alternate-day regime.